and was always regular and painless. She had given birth to a child that died at the age of four months without apparently having been ill. There had been no miscarriage. There was nothing in the family history having any bearing on the patient's illness.

While the total absence of sensibility of all forms in the right upper extremity suggests the possible presence of hysteria, the lack of tactile sense, especially when taken in connection with the exaggeration of the reflexes in the lower extremities, does not preclude the diagnosis of syringomyelia.

Dr. Mills said it is possible to have syringomyelia of traumatic origin but it is uncommon. Dr. Mills alluded to a case at the University Hospital which appeared to be of that nature, but it seemed to him that the case of Dr. Eshner could be explained on the theory of a hematomyelia, especially because of the suddenness.

A CASE OF PROGRESSIVE MUSCULAR ATROPHY WITH REMARKABLE IMPROVEMENT

By J. K. Mitchell, M.D.

The interest of the patient presented lies in two points—the diagnosis and the unusually good results of treatment. The history is briefly as follows: G. S., æt. 24 years, an automobile mechanic, has no points of interest in the personal or family history up to the beginning of the condition from which he now suffers, except scarlet fever in his ninth year, during which he is said to have had meningeal symptoms. Not long after some deformity or weakness of the left foot was noticed and an increasing symmetrical atrophy all over the body developed slowly from that time. At 18 years the little toe of the left foot was frost-bitten and had to be amputated, and, although it healed, an open ulcer afterwards developed and has never closed.

He has been at work until he came to the Orthopedic Hospital for treatment of this ulcer and was referred from the surgical clinic to the neurological service for care of his atrophic trouble.

He has, very distinct, general wasting, affecting all his muscles except those of the face. Sensation in all forms and everywhere is normal except on the toes, where it is slightly diminished to touch. The knee-jerk is lost, the skin reflexes are present, there are no abnormal reflexes. The atrophy of the foot muscles has resulted in a slight pes cavus and this, with his weakness, causes a swaying gait, but his station is perfect. He has no pain or tenderness anywhere. Electric examination of the muscles developed the interesting fact that there was no reaction of degeneration, but an evenly distributed quantitative reduction to both galvanism and faradism, not of very high degree.

The question of diagnosis is interesting here. At first polyneuritis seemed most likely in view of the history of infectious disease and of the legs having shown the earliest signs of trouble, as is usual in infectious polyneuritis. The continuation and slow spread of the trouble without pain, seem to render this improbable. The character of the onset, the subsequent history, and the electric contractility also exclude the possibility of chronic poliomyelitis. Nor can we think it amyotrophic lateral sclerosis in view of the absence of any involvement of the pyramidal tracts, usually found degenerated in that disorder. Simple weakness, widely disseminated progressive wasting, with loss of deep reflexes in the wasted parts, force on us the conclusion of progressive muscular atrophy.

Whatever the diagnosis, improvement to any great degree in any of the forms of neuritic or spinal atrophy after a duration of sixteen years, is unusual enough to be worth recording. S. weighed 92 pounds on admission to the Orthopedic Hospital in May, 1908. He has gained 18 pounds and this increase in weight is not fat but muscle. We have all seen patients with the various atrophies get fat in bed and lose strength at the same time, but this man has improved in activity and endurance. When he came to the hospital he could walk a few hundred feet with difficulty and fatigue, although he could do his work and move about a workshop. In October he had leave of absence from the hospital to see the "Founders' Week" celebration and announced with pride on his return that he had walked five miles without difficulty. All his muscles have increased in bulk and have lost the harsh, dry feeling they had when palpated at the time of his admission.

He was kept in bed at first with persistent massage and faradism and later was up and about the wards but had no coördination exercises, or, as we prefer to call them at the Orthopedic, "precision movements" until the past few weeks, so that his improvement is not merely that he uses his muscles more accurately, but is a real gain in power.

Dr. W. G. Spiller hesitated to accept the diagnosis of progressive spinal muscular atrophy, a disease in which the cells of the anterior horns are primarily involved, as the improvement was said to be so great. Sharp distinction must be made between primary and secondary changes of the anterior cornual cells, for while it is not uncommon to find alteration of these cells as a part of multiple neuritis, the alteration is of a different type from that of progressive spinal muscular atrophy or amyotrophic lateral sclerosis, and the prognosis of recovery is very different. The whole neurone is likely to be affected in a long-standing process, but we must continue to make distinctions between the cellular changes that occur from some cause directly affecting the cell body, and one affecting the distal end of the neurone. The prognosis of multiple neuritis is good as a rule, but the prognosis of spinal atrophy is grave. The microscope reveals also a very different type of cellular change in the two forms. It is the general opinion that when the anterior cornual cells are severely *primarily* degenerated they do not recover.

Dr. Spiller thought the case was probably one of motor neuritis, and he referred to a similar case with necropsy reported some thirteen years ago by Dejerine and Sottas.[1]

Dr. F. X. Dercum thought the really remarkable thing about the case was the degree of improvement following the atrophy, whatever its cause, whether the disease was limited to the spinal neurones or involves also their axones. The man had a distinctly steppage gait; he raised his feet and threw them forward as do cases of peripheral neuritis. The thought also suggested itself that possibly the case was one of primary neurotic atrophy, but Dr. Dercum thought that this diagnosis could be discarded because of the history. He thought Dr. Mitchell's inference that the weight had been due to actual gain in muscle correct, and that to him is remarkable. He asked Dr. Mitchell when treatment was begun in the case and how long it was carried out.

Dr. C. W. Burr said that it seemed very possible to him that the improvement in the man had been due to the education he had gotten, that is since he has been under Dr. Mitchell's care, the education in the use of what muscles he has. He had been taught precision in movement and certain gymnastic exercises, and it is certainly quite possible that while there had been no real anatomical improvement in his condition that he now can do things with his muscles that he could not do.

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[1] Comptes rendus heb. des séances de la Soc. de Biol., 1896, p. 193.

Dr. D. J. McCarthy thought the history of the case (the condition developing after scarlet fever) might throw some light on differentiating the condition from one of a progressive muscular atrophy, types of slow onset. Dr. McCarthy said he had had at different times two cases which have had some similarity to these present cases. The first was a case which he examined on two or three occasions, extending over a period of three or four weeks, and then the man went to Dr. Thomas, of Baltimore. After a stay at Johns Hopkins Hospital the symptoms cleared up entirely. The man was about 17 or 18. He was on an automobile trip to Atlantic City; it was a very hot day. He was very tired from perspiration, and the following day developed the condition for which Dr. McCarthy was consulted. He had no pain, simply muscular tremors. When Dr. McCarthy examined him he had the appearance of a case of muscular atrophy, marked loss of power in the four extremities, marked fibrillary tremor, affecting practically all the muscles of the four extremities. Dr. McCarthy saw him a week after the condition developed and the symptoms continued at first under Dr. Thomas's care, but later disappeared with some loss of power, but lack of evidence of progressive change in the muscles themselves. Of course, that case is not so remarkable as the one Dr. Mitchell presents. In that case there was no sensory disturbance. That case cleared up, or began to clear up, evidently within the period of time of regeneration of the ganglion cells; that is, in the period of twenty to thirty days.

The other case was a man of 40, who suffered from some type of infection. He worked at Point Breeze Oil Works; he was in bed three weeks with a fever and at the present time has a muscular atrophy which at the end of a year shows no improvement. It developed suddenly with fever, and presented all the clinical picture of a case of progressive muscular atrophy. At the present time he shows the symptoms of progressive muscular atrophy.

Dr. McCarthy thought these two cases were cases in which the condition was not isolated entirely to the anterior horn cells, but a subacute change which affects the entire peripheral neurone. When a partially degenerated nerve is put under conditions offering a chance for restitution there is no reason why restitution of function should not take place. In many of the organic conditions, more particularly in tabes, changes from the normal type of structure occur in the anterior horn cells, and restitution of function is possible, provided the cause is removed. Dr. McCarthy believes anyone who studies the nerve cells in a large number of cases comes across, not infrequently, partial loss of function or destruction, without complete loss of structure, and there is no reason why this partial degeneration, without complete loss of function, or complete loss of structure, should not exist for a long period of time.

Dr. J. K. Mitchell, in closing, said he thought that what Dr. McCarthy had said answered Dr. Spiller's objection. There seems to be a possibility in this case of three diagnoses, any one of which will serve our purpose as long as the patient is getting better, in which the symptoms do not differ very radically, the type symptoms being a little uncertain in all. The improvement reminds one of what we see in old poliomyelitis anterior. Patients come in, 20 or 25 years of age, who have had no treatment since they had the disease when three or four years old, and it looks as if there was total paralysis. When you wake up these cases by treatment you find some muscles which after all were not involved but have only been neglected and are suffering because they have not been used. Electricity thoroughly applied, massage and proper feeding, brings them up. Whether it is a question of muscles, or whether it is a question of getting the best out of partially injured or little injured cells in the cord, is also to be considered and one simply cannot tell unless we have a post mortem both before and after diagnosis.

As to the question of treatment, the man has been under treatment since the first of June, when he was put to bed and kept entirely at rest, the theory being that the remaining nerve cells in the cord were overburdened like an electric wire already overloaded and asked to carry more current, with, as a consequence, chronic exhaustion, so that rest, not for the muscles, but for the overtasked nerve cells, was the thing that was going to help in the first place and then afterward we could proceed with more active treatment. In muscles in which there seemed almost no substance left we could get a normal reaction, though very much reduced, but not a reaction of degeneration. Faradism was kept up steadily and the quantity and quality of reaction distinctly improved. After two or three months of this when he began to go about, he was put on exercises of precision. It should be noted that the man was working until the time of his coming into the hospital as an automobile mechanic, therefore he was getting plenty of ordinary exercise, and was able to make use of his muscles, though less well than now.

Dr. C. K. Mills presented a patient and made some remarks on the differential diagnosis of grave hysteria and organic disease of the brain and spinal cord, especially disease of the parietal lobe. (*To be published in this journal.*)

Dr. Dercum thought the Babinski reaction undoubtedly present; it was not the pronounced Babinski that we see in the typical organic case, but it was none the less present. He regarded the case as organic. There were no hysterical stigmata and not even the man's mental attitude was one of hysteria. In multiple cerebro-spinal sclerosis, it is not uncommon for sudden attacks, suggesting apoplectiform attacks to be present early in the history; it is not infrequent also to receive a history of early improvement or such a case may suggest a functional disease at first but which later on proves to be organic.

Dr. Mills queried whether it might not be a case in the early stage of paresis.

Dr. Dercum replied it was perfectly possible, and asked whether Dr. Mills had elicited a history of syphilis.

Dr. Mills replied that he thought the man had syphilis.

Dr. Dercum said perhaps it was paresis in the early stage. He did not, however, have that slight mental obtuseness, that slight mental haze present even early and which is indicative of an active and close contact with the outside world. The patient seemed quite alive and alert in his responses.

Dr. Burr said that from the superficial examination he was able to make of the man's mental state he was inclined to think that the patient might be a case of beginning paresis. In paresis it is not very infrequent for patients to be somewhat hysterical in manner.

Dr. McCarthy thought the discussion ought not to be closed without saying a word as to the Babinski sign. There was extension of the toes.

There was flexion of the toes on deep irritation and then the quick response on slight irritation to which there seemed some objection. While it is perfectly true in the Babinski sign there is a slow response, in others there is a quick response which is quite as positive as if it were deliberate and slow. Dr. McCarthy thought the great value of the Gordon phenomenon is in the cases where you are in doubt as to whether you are dealing with the Babinski sign or not. That is where he has found Oppenheim's and Gordon's sign to be of value. Where these phenomena are absolute and positive, then he considered them to be in favor of a Babinski reflex.

Dr. Eshner said that it is a common observation that patients suffering from multiple sclerosis often exhibit a certain optimism, cheerfulness and hopefulness even while the symptoms and physical signs are growing progressively worse.

TWO CASES OF ASTASIA-ABASIA

By F. X. Dercum, M.D.

The histories are in abstract as follows:

Case I.—A. M., age 19, unmarried; family history negative; personal history negative save an attack of measles at two years of age, which was followed by weakness of both legs, persisting for six months. Two years ago patient suffered from an attack of typhoid fever which confined her to bed for four weeks. For fifteen weeks following she could not stand upon her feet. Subsequently she began to walk with a staggering, swinging gait which slightly improved. A severe fright caused a sudden recrudescence of the symptoms and also brought on hysterical crises.

Present Condition.—Is well nourished but rather obese. Has a high color. Exposed to the air, the skin over the chest assumes a mottled pink-and-white appearance. Dermography also is marked. Examination discloses markedly impaired station and very ataxic gait. There is no paralysis and no atrophy. Reflexes present no anomalies save that both knee-jerks are plus. The general visceral examination is negative. There are no sensory losses. There are, however, typical areas of painful hyperesthesia under the left breast, and over both groins and over the mid-scapular and lumbar spines and over the sacral and coccygeal regions.

Examination of the eyes reveals no abnormalities save that the fields are slightly contracted and sharply indented for red and that the color fields are at places reversed.

The gait which is the most striking symptom presented by the patient presents the following peculiarities.

The patient cannot rise steadily from the sitting posture. When finally she gets on her feet, she is able to walk, but her gait is slow, labored and absolutely unique. One side of the pelvis is swung forward, and with it the leg. The body lurches forward and after swaying for twenty to thirty seconds, she balances herself on the leg. The body is bent forward and the eyes are fixed to the ground. It seems that in her oscillations she would surely fall. The opposite leg is now brought alongside the first and again the body sways. The feet are planted rather widely apart and as she stands the weight is pivoted on the heels, while the balls of the feet rise and fall alternately from the ground. The oscillations of her trunk, both as she stands and as she attempts to walk strongly resemble the titubation seen in cerebellar disease.

Case II.—M. C., woman, age 28, unmarried. Family history negative